The Electrocardiogram in Children with Ventricular Septal Defect and Severe Pulmonary Hypertension

Correlation with Response of Pulmonary Arterial Pressure to Surgical Repair

By James W. DuShane, M.D., William H. Weidman, M.D., Robert O. Brandenburg, M.D., and John W. Kirklin, M.D.

SUCCESS in the surgical treatment of children who have large ventricular septal defects is dependent in part on the proper selection of patients for operation. The patients most deserving of such correction are those who have severe pulmonary hypertension, and it is in these that the decision to offer surgical intervention may present the greatest problems.

The factors influencing the increase in pulmonary arterial pressure deserve critical analysis. Elevated pulmonary arterial pressure in patients with large ventricular septal defects is, to a great extent, the result of 2 factors, namely increased pulmonary blood flow or increased pulmonary vascular resistance; both factors are present in some instances. In those patients without pulmonary vascular disease but with a high pulmonary blood flow in relationship to systemic blood flow and with only slightly increased pulmonary vascular resistance, the pulmonary arterial pressure may be equal or nearly equal to the systemic pressure. In those who have complicating pulmonary vascular disease in whom the pulmonary resistance is increased and the pulmonary blood flow is not increased in relationship to systemic flow, the pulmonary and systemic pressures may be equal or nearly equal.

If the increased pulmonary arterial pressure is primarily the result of increased pulmonary blood flow from a left-to-right shunt, surgical closure of the defect will result in a decrease in pulmonary arterial pressure. If the increased pulmonary pressure is secondary toobliterative changes in the pulmonary vasculature, with the pulmonary blood flow only equal to or less than the systemic flow, the shunt being predominantly from right to left, then closure of the defect will not result in an immediate decrease in pulmonary arterial pressure. In the latter situation, operative repair of the defect is thought to be detrimental to the patient and probably should be avoided.

The important consideration in the choice of patients to whom surgical treatment should be offered is the relationship between pulmonary and systemic blood flows, whether the shunt is predominantly from left to right or right to left, rather than the height of the pulmonary arterial pressure by itself. The present study is concerned with the evaluation of this relationship between pulmonary and systemic blood flows from electrocardiographic data.

When the dominant shunt is from left to right and the pulmonary blood flow is greater than the systemic flow, the extra volume of shunted blood returns to the left side of the heart, resulting in an increase in the work of the left ventricle. When the shunts are balanced or dominantly from right to left, there is no increase in pulmonary blood flow or in the work of the left ventricle. The electrocardiogram, when properly interpreted, reflects increased work by the left ventricle. Thus, electrocardiographic evidence of left ventricular overloading in patients with isolated ventricular septal defect who do not have mitral or aortic valvular disease, coartation of the aorta, or systemic hypertension should indicate...
a left-to-right shunt and an increased pulmonary blood flow regardless of the level of pulmonary arterial pressure.

Methods

Electrocardiograms from 100 children with ventricular septal defects whose pulmonary arterial pressures were 80 to 100 per cent of systemic pressures and who had been operated on were mixed with 25 electrocardiograms from children without demonstrable cardiac disease and with 17 electrocardiograms from children who had ventricular septal defects and pulmonary hypertension whose shunts were proved to be dominantly from right to left. These tracings were analyzed by the first 3 authors independently, after all identifying information had been removed except the age of the child, in an effort to determine electrocardiographic evidence of left ventricular overwork.

Specific standards for electrocardiographic signs of an overworking left ventricle in infants and children have not been established, particularly when coexistent patterns of right ventricular hypertrophy are present. A number of features of the scalar electrocardiogram suggest left ventricular overwork, but many of these are difficult to define clearly or do not lend themselves to analysis by numerical limits or ratios. Seven criteria for left ventricular overwork were chosen arbitrarily for this study, since these criteria can be defined clearly and applied by any investigator. It is emphasized that these are not absolute and infallible. Some normal infants and children may have some of these features, although such instances are rare.

Left ventricular overwork was considered to exist if one or more of these 7 electrocardiographic features were present:

1. Peaked, symmetric, or nearly symmetric T waves in leads reflecting left ventricular potential in children at any age (lead V6, or leads II, III, and aVF) (criterion 1).
2. Q waves greater than 0.4 mV (4.0 mm.) in precordial lead V6 in patients more than 3 years of age.
3. R waves greater than 2.5 mV (25 mm.) in precordial lead V4 in children at any age.
4. S waves greater than the R waves in lead V3R or V1 in patients less than 2 years of age, or S waves in lead V1 greater than 2.5 mV (25 mm.) at any age.
5. Axis of mean QRS vectors in the frontal plane of +60 degrees or less in infants less than 3 years of age (fig. 2).
6. Counterclockwise QRS loops in the frontal plane, as determined by vectorial analysis of the scalar electrocardiograms, in infants less than 3 years of age (fig. 3).
7. Combination of criteria 5 and 6 in children less than 6 years of age (fig. 3).

The criteria (1, 5, 6, and 7) that are not mathematically definable are illustrated in the afore-mentioned 3 figures. The pulmonary arterial and systemic pressures in the 3 patients from whom these

*Circulation, Volume XXII, July 1960*
electrocardiograms were obtained are summarized in table 1. Pulmonary arterial and systemic arterial pressures had been measured in the operating room immediately before and after surgical repair of the defects. The response of the pressures to closure of the defects was compared with the electrocardiographic evidence of left ventricular overwork in each case. A decrease in the pulmonary arterial systolic pressure to a level of 70 per cent or less of systemic arterial systolic pressure was considered to be significant evidence of pre-existing increased pulmonary flow. A decrease to 90 per cent was considered insignificant. A decrease to 75 or 80 per cent of the systemic pressure was studied carefully and was considered to be significant if the pulmonary arterial pressure was actually equal to (100 per cent of) the systemic pressure before operation in that particular patient, thus indicating a pulmonary flow a little in excess of the systemic flow.

Results

All the electrocardiograms from the 25 children without demonstrable cardiac disease were considered to be normal by the authors. All the electrocardiograms from the 17 patients who had obvious, predominant right-to-left shunts and cyanosis were considered to show evidence of increased pressure in the right ventricle, but none was considered to demonstrate overwork of the left ventricle.

The electrocardiograms from 90 of the 100 patients with ventricular septal defects and pulmonary hypertension who had surgical closure of the defects showed evidence of left ventricular overwork; all of these 100 patients also had electrocardiographic evidence of increased right ventricular pressure. The pulmonary arterial systolic pressure in 89 of these 90 patients decreased after repair, giving a correlation of 99 per cent and proving a pre-existing pulmonary blood flow. In these 89 cases, the pulmonary arterial pressure in 1 decreased to 75 per cent of the systemic pressure, but the preoperative pulmonary pressure had been 100 per cent of the systemic pressure in this instance; the pulmonary arterial pressure decreased to 70 per cent in 2 cases, and in the remainder it decreased to a level of 60 per cent or less of the systemic pressure.

The remaining 10 of the 100 patients who had ventricular septal defects and pulmonary hypertension were considered not to exhibit any of the electrocardiographic criteria for left ventricular overwork already listed. The pulmonary arterial pressure did not decrease significantly in 5 of these after repair, indicating the absence of a preexisting increased pulmonary blood flow. Of the remaining 5 patients, 1 had a postoperative pulmonary pressure that was 75 per cent of the systemic pressure, but the preoperative pulmonary pressure had been 80 per cent of the systemic pressure, indicating no significant increase in pulmonary flow; 2 patients had a postoperative pulmonary systolic pressure of 75 per cent of the systemic pressure as compared with a preoperative level of 100 per cent, thus indicating a slight increase in pulmonary blood flow; the remaining 2 patients experienced a significant decrease in pulmonary arterial pressure, the final level being 70 per cent of the systemic pressure or less.

Discussion

If a ventricular septal defect is large, there is little resistance to the flow of blood across the defect. The relationship of pulmonary arterial resistance and systemic arterial resistance is, therefore, a dominant factor in determining the relative amounts of blood flowing
from the ventricles into the pulmonary artery and aorta. In the absence of obstructive disease in the pulmonary vasculature, the pulmonary resistance is less than the systemic resistance and the pulmonary blood flow is greater than the systemic flow. The hemodynamic continuity of the left ventricle and the pulmonary artery is reflected by the presence of systemic, or near systemic, pressure in the pulmonary artery. When severe pulmonary vascular obstructive changes occur, the pulmonary resistance may approach or equal the systemic resistance, with resultant equalization of pulmonary and systemic blood flows. The pulmonary arterial pressure continues to reflect the pressure in the ventricles.

The proper selection of patients for closure of ventricular septal defects cannot be judged by the mortality rate alone, since deaths may be associated with a complicating heart block, faulty technic in extracorporeal circulation, failure to close the defect completely and other technical surgical factors, as well as imperfect postoperative care. The success of the operation is determined in large part by the return of the circulation to normal or near to normal after closure of the defect. The persistence of severe pulmonary hypertension months after repair of a ventricular septal defect raises doubt as to the success of the operation.

The preoperative relationship of pulmonary arterial pressure to systemic pressure is in itself an unimportant criterion for the selection of patients for surgical intervention; the response of the pulmonary arterial pressure to repair is the important consideration. If the pulmonary flow does not exceed the systemic flow, an unsatisfactory response to closure of the defect is likely, and surgical treatment should not be offered.

Pulmonary and systemic blood flows may be calculated from the data obtained during cardiac catheterization. These data reflect the hemodynamic situation existing during the time of cardiac catheterization and may be influenced by artificial circumstances attending the procedure. Anesthesia or sedation, with resultant changes in pulmonary vascular resistance, pulmonary ventilation, and systemic oxygen saturation, may interfere with accurate interpretation of the data. Catheterization of a child without anesthesia may result in an unsteady physiologic state and may invalidate the formulas used for calculating systemic and pulmonary blood flows.

Evidence of increased left ventricular work in the electrocardiogram, with its concomitant increased pulmonary blood flow in a patient with ventricular septal defect should reflect the status of the shunts existing in the patient under those conditions present in his day-to-day living. Nadas has not supported the views of Cabrera and Monroy and has been unable to diagnose overloading of the left ventricle using their criteria. Fyler and associates stressed the necessity of cardiac catheterization to define the size of the left-to-right shunt and the level of pulmonary vascular resistance in those patients who present electrocardiographic evidence of combined ventricular hypertrophy. Dreifuss and co-workers, Zacharioudakis and associates, and Char's group

Circulation, Volume XXII, July 1960
analyzed the electrocardiogram in cases of ventricular septal defect, measuring the voltage of the R, S, and T waves, and calculating values of R/S ratios; they found little or no correlation of the electrocardiogram with pulmonary blood flow, pulmonary pathologic changes, or surgical mortality rates.

The entire electrocardiogram must be studied carefully in searching for signs of left ventricular overwork. In some instances, lead V₆ may not be representative of the left ventricular potential, and one must examine leads II, III, and aVF in some cases and leads I and aVL or V₃ in others to assess the status of this chamber. The only evidence of left ventricular overloading in patients less than 3 years of age may be an axis deviation of the mean QRS vectors of +60 degrees or less or the presence of a counterclockwise QRS loop in the frontal plane, determined by vectorial analysis of the scalar electrocardiogram, features that are rarely seen in normal infants. Occasionally, Q waves greater than 0.4 mv. (4 mm.) are present in one or more of leads II, III, aVF, and V₆ in infants less than 3 years of age without demonstrable cardiac disease, and this change may not, in the absence of other criteria, indicate overwork of the left ventricle.

The best proof of increased pulmonary blood flow is a decrease in pulmonary arterial pressure in relationship to systemic pressure immediately after the ventricular septal defect is closed. A comparison of electrocardiographic evidence of left ventricular overwork with the response of pulmonary arterial pressure after closure of the defect proved the validity of electrocardiographic interpretation of left ventricular overwork, inasmuch as 89 of the 90 patients with such evidence exhibited proof of increased pulmonary flow. The fact that 4 of the 10 patients not showing electrocardiographic evidence of left ventricular overwork had a slight to a moderate decrease in pulmonary pressure indicates that all information obtainable, whether clinical or physiologic, should be employed in selecting such patients for operation. We currently use this type of evaluation in clinical practice. Patterns of right ventricular hypertrophy were present in the electrocardiograms from all patients with ventricular septal defect in this study. Such patterns reflect pulmonary hypertension regardless of cause, whether primarily from increased pulmonary flow or from obliterator changes in the pulmonary vessels; thus, they are of no prognostic significance except as related to the associated presence or absence of evidence of left ventricular overwork.

**Conclusions**

The height of the pulmonary arterial pressure is in itself of no prognostic importance in determining which patients will have a successful response to surgical closure of a ventricular septal defect.

Of prime importance in the selection of patients for this operation is the relationship of the volume of blood flowing through the pulmonary and the systemic circuits. Those patients whose pulmonary blood flow is not in excess of the systemic flow, and who thus have a dominant right-to-left or a balanced shunt, will not experience a reduction in pulmonary arterial pressure after repair.

Electrocardiographic evidence of increased left ventricular work has proved to be of great value in assessing the pulmonary blood flow, regardless of the presence of severe pulmonary hypertension. Patterns of right ventricular hypertrophy in the electrocardiogram usually do not mask evidence of left ventricular overwork when the criteria suggested in this paper are applied, as evidenced by the 99 per cent correlation between electrocardiographic evidence of increased left ventricular work and proof of preexisting increased pulmonary flow in the 90 surgically treated patients with ventricular septal defect and severe pulmonary hypertension fulfilling such criteria.

On the basis of these observations, it is believed that the electrocardiogram has an important role in the selection of such patients for surgical treatment. In the absence of electrocardiographic evidence of left ventricular overwork, no patient should be denied surgical intervention if evidence for increased pulmonary flow can be obtained by any other means.
**Summario in Interlingua**

Le nivello del tension pulmono-arterial per se ha nulle importantia prognostic in determinar qual patientes respondera favorabilmente al clausion chirurgie de un defecto ventriculo-septal.

De prime importantia in le selection de patientes pro iste operation es le relation del volumine de sanguine que passa per le circuitos pulmonar e sistemic. Patientes in qui le fluxo pulmonar non excede le fluxo systemic e in qui, per consequente, le shunting es dominantemente dextero-sinistre o balanciate non experienciara un reduction del tension pulmono-arterial post le reparo chirurgie.

Evidentia electrocardiographic de un augmento del travaillo dextero-ventricular se ha provate de grande valor in le evaluation del fluxo de sanguine pulmonar sin reguardo al presentia de sever hypertension pulmonar. Configurationes electrocardiographie correspondente a hypertrophia dextero-ventricular non oculta le evidentia de excesso de travaillo sinistro-ventricular si le criterios proponite in le presente articulo es applicate. Iste assertion pare esser justificate, viste le correlation de 99 pro cento inter le indicationes electrocardiographic del augmento del trabalho sinistro-ventricular e le prova de un pre-existent augmento del fluxo pulmonar in le 90 chirurgicamente tractate patientes con defecto ventriculo-septal e hypertension pulmonar sever ubi le mentionate criterios esseva observe.

Super le base de iste observationes le opinion pare justificate que le electrocardiogramma ha un rolo importante in le selection, inter iste patientes, del cases appropriate al trattamento chirurgie. In le absentia de provas electrocardiographic de excessos de trabalho sinistro-ventricular, nulle patiente debera perder le beneficios del intervention chirurgie si le prova de un augmento del fluxo pulmonar pote esser obtenite per un altere methodo.

**References**


So great was this Greek contribution to medicine that it is by no whim of chance that our medical terminology is so largely Greek in origin, that we call the essential method of medical reasoning the Hippocratic method, and that as heirs to so remarkable a heritage we choose to bind ourselves with the Hippocratic oath. For, following the Greeks, we now take disease to be an entirely reasonable process, obeying laws eventually patent to observation and to reasoning. There are exceptions, perhaps, to so categorical a eulogy of the Greek interpretation of the nature of disease. I doubt, for example, whether the Greeks maintained quite so detached, rational, and naturalistic a concept of mental diseases or epilepsy. But even if the Greeks had managed to be to some extent rational about insanity, the demoniac possession theories prevailed in later centuries. But in the main, the immense advantage of the Greek view was that, in the relative absence of superstition and fear, it built, with its amazingly rational approach, at least the scaffolding for the advancement of knowledge.—ALAN GREGG, M.D. Challenges to Contemporary Medicine. New York, Columbia University Press, 1956, p. 32.
The Electrocardiogram in Children with Ventricular Septal Defect and Severe Pulmonary Hypertension: Correlation with Response of Pulmonary Arterial Pressure to Surgical Repair

JAMES W. DUSHANE, WILLIAM H. WEIDMAN, ROBERT O. BRANDENBURG and JOHN W. KIRKLIN

Circulation. 1960;22:49-54
doi: 10.1161/01.CIR.22.1.49

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1960 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/22/1/49.citation

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/